RESEARCH ARTICLE DOI: 10.53555/jptcp.v30i17.2628

"TO COMPARE DIFFERENT PARAMETER OF ABG AND VBG IN PATIENTS WITH KETOACIDOSIS CASES OF DIABETES"

Dr Ragini Tarole^{1*}, Dr Pradeep Prajapati², Dr Divya³, Dr Seema⁴

^{1*}junior resident, Department of medicine, GRMC Gwalior
²Associate professor, Department of medicine, GRMC Gwalior
³junior resident, Department of medicine, GRMC Gwalior
⁴Senior resident, Department of medicine, SSMC Rewa

*Corresponding Author: Dr Seema

*4Senior resident, Department of medicine, SSMC Rewa

Abstract-

Introduction- Diabetes mellitus is common endocrinopathy. It is a group of common metabolic disorder which share the phenotype of hyperglycemia. DKA is acute, severe disorder directly related to diabetes. DKA was formerly considered a hallmark of type 1 DM, but it also occurs in individuals with type 2 DM who can sometimes subsequently be treated with oral glucose-lowering agents. Performing a VBG rather than an ABG is particularly convenient in the ICU and in the emergency department, either peripherally or from a central venous line from which venous blood can be quickly drawn.

Aims and objectives- This is a prospective study, was conducted in Department of General Medicine, G.R. Medical College, Gwalior (M.P.) from Jan 2021 – Jun 2022. The main aim of the study is"To compare different parameter of ABG and VBG in patients with ketoacidosis cases of diabetes".

Methods and materials- This is a single center observational study. In this study, demographic, clinical and laboratory details were studied in patients presenting with diabetic ketoacidosis satisfying the study population criteria and the impact on the outcome was assessed. 96 patients who will be getting admitted in medical units at Jayarogya hospital and group were randomly selected as per the inclusion and exclusion criteria. Written informed consent will be obtained from each diabetic patients enrolled in the study.

All the dates will be entered in a data collection sheet in an Excel format and analysed using SPSS Software. Numerical values will be reported using mean and standard deviation or median. Categorical values will be reported using number and percentages. Probability value (p) value less than 0.05 was considered a statistically significant.

Results- Among the studied 97 patients, maximum cases belonged to the age group 41-60 years (n=34). Mean age of the study population was 40.53 years with standard deviation of 17.47 years. Out of studied 97 patients, 70 were male and 27 were female. In our study, most common clinical symptom was vomiting (found in 28.9% patients), followed by altered sensorium (24.7% each), while abdominal pain 22.7% and shortness of breath was found in 17.5 % patients respectively. Among the studied 97 patients, 16 were having associated hypertension and 4 patients were having CAD, while

only 2 patients were suffering from hypothyroidism. In our study, Type 1 DM patients were 43.3% while 56.7% were Type 2 DM. Among the patients 41.2% shown no ECG changes, while 50.5% shown sinus tachycardia and only 8.2% shown significant ECG changes with tall T waves in precordial leads. Among the studied 97 patients, 19.6% expired while 80.4% recovered. The mean value of Arterial pH is 7.24±0.07 and Arterial bicarbonates is 18.74±2.73. while mean Venous pH is 7.26±0.06 and Venous Bicarbonates is 19.58±2.67 in expired patients.

The mean value of Arterial pH is 7.25 ± 0.07 and Arterial bicarbonates is 17.07 ± 3.06 while mean Venous pH is 7.25 ± 0.06 and Venous Bicarbonates is 18.07 ± 3.04 in expired patients.

Conclusions- It was concluded that venous blood gas analysis has got advantages over arterial blood gas analysis like safety, fewer number of punctures, easy sampling, less painful, less invasive even though there are some reservations' analysis safer alternative to ABG for determining acid base status reducing the need for frequent invasive arterial sampling.

This study suggests that VBG pH values very closely correlate with ABG pH values, which also shows VBG substitution for ABG.

Hence venous blood gas might be used as an ideal alternative to arterial blood gas in the initial management of patients in Diabetic Ketoacidosis.

Keywords- arterial blood gas, diabetic ketoacidosis.

INTRODUCTION

Diabetes mellitus is common endocrinopathy. It is a group of common metabolic disorder which share the phenotype of hyperglycemia. **It** is a group of chronic non-communicable disorders characterized by increased plasma glucose levels above defined normal limits. It is associated with a relative or absolute impairment in insulin secretion, along with varying degrees of peripheral resistance to the action of insulin [1].

Classic symptoms of hyperglycemia include polyuria, polydipsia, nocturia, blurred vision, and, infrequently, weight loss. These symptoms are often noted only in retrospect after a blood glucose value has been shown to be elevated. Polyuria occurs when the serum glucose concentration rises significantly above 180 mg/dL leading to glycosuria causing osmotic diuresis, which in turn can lead to polydipsia.

The adults with DM can also present with features of hyperosmolar hyperglycemic state or diabetic ketoacidosis.

Criteria of diagnosis of diabetes mellitus (AMERICAN DIABETES ASSOCIATION)

- Fasting blood sugar >/=126mg/dl
- Post prandial blood sugar >/= 200mg/dl
- HBA1C >/= 6.5%
- RBS>/= 200mg/dl with symptoms

DKA is acute, severe disorder directly related to diabetes. DKA was formerly considered a hallmark of type 1 DM, but it also occurs in individuals with type 2 DM who can sometimes subsequently be treated with oral glucose-lowering agents (frequently in individuals of Hispanic or African-American descent). DKA is associated with absolute or relative insulin deficiency, volume depletion, and acid-base abnormalities.

Pathophysiology of DKA results from relative or absolute insulin deficiency combined with counterregulatory hormone excess (glucagon, catecholamines). Both insulin deficiency and glucagon excess, in particular, are necessary for DKA to develop. The decreased ratio of insulin to glucagon promotes gluconeogenesis, glycogenolysis, and ketone body formation in the liver, and increases in substrate delivery from fat and muscle (free fatty acids, amino acids) to the liver. Ketosis results from markedly increased FFA release from adipocyte. Reduced insulin levels, in combination with

elevations in catecholamines and growth hormone, also increase lipolysis and the release of free fatty acids. Markers of inflammation (cytokines, C-reactive protein) are elevated in DKA

Signs and symptoms of diabetic ketoacidosis Common symptoms of DKA are polydipsia and polyuria.

An arterial blood gas (ABG) is the traditional method of estimating the systemic carbon dioxide and pH, usually for the purpose pf assessing ventilation and or acid base status.

There are two ways to analyses blood gases

- Arterial blood gas analysis
- Venous blood gas analysis

The arterial blood sample can be difficult to take due to diminished pulses or patient movement example people in shock. Diminished pulse can reflect poor peripheral circulation or low blood pressure, while patient movement is caused by the pain associated with arterial puncture. A venous blood gas (VBG) is an alternative method of estimating systemic CO2 and pH that does not require arterial blood sampling. Performing a VBG rather than an ABG is particularly convenient in the ICU and in the emergency department, either peripherally or from a central venous line from which venous blood can be quickly drawn.

AIMS AND OBJECTIVES

This is a prospective study, was conducted in Department of General Medicine,

G.R. Medical College, Gwalior (M.P.) from Jan 2021 – Jun 2022. The main aim of the study is-

"To compare different parameter of ABG and VBG in patients with ketoacidosis cases of diabetes".

MATERIALS AND METHODS

STUDY DESIGN- This is a single center observational study. In this study, demographic, clinical and laboratory details were studied in patients presenting with diabetic ketoacidosis satisfying the study population criteria and the impact on the outcome was assessed. Sample size: 96 patients

STUDY POPULATION: 96 patients who will be getting admitted in Medical units at Jayarogya hospital and group were randomly selected as per the inclusion and exclusion criteria.. Written informed consent will be obtained from each diabetic patients enrolled in the study.

INCLUSION CRITERIA: All diagnosed adult diabetic patients in diabetic ketoacidosis who will be admitted in Medical units at Jayarogya hospital and group.

EXCLUSION CRITERIA:

- Patients with diagnosed systemic causes of renal disease, chronic diarrhoea.
- Pregnant women and children age less than 18 years
- Patients having preexisting respiratory illness.

Clinical assessment and demographic details: The initial clinical details on arrival at emergency department which included symptoms at presentation, vital signs, neurological status were assessed based on Glasgow coma Scale (GCS) and other specific system findings were collected from the case records. Then after establishment of diagnosis of diabetic ketoacidosis demographic history and thorough clinical details were collected from the patient and /or the closest relative.

Laboratory assessment (DKA patients): After the initial clinical assessment, blood investigations were taken, which included random blood sugar (RBS), serum electrolytes, renal function tests (RFT), liver function tests (LFT), plasma acetone, arterial blood gas analysis (ABG), complete blood count (CBC). In addition to lipid profile, glycated haemoglobin (HbA1c), urine routine examination and urine acetone were done for all patients included in the study. Other additional investigations

which were done included serum amylase, serum lipase, C-Reactive protein (CRP), chest x-ray, blood culture and sensitivity and urine culture and sensitivity based on individual patient requirements.

DATA COLLECTION

96 diagnosed diabetic patients in DKA will be included in the study. Detailed history – including duration of disease, any past history of and other medications, treatment regimen started. Patients will be examined in detailed for assessing any symptoms and signs of Diabetic ketoacidosis. Blood samples will be taken for ABG and VBG allen test is performed

- 1. Instruct patient to clench his fist.
- 2 using occlusive pressure to both ulnar and radial arteries to obstruct blood flow to the hand
- 3. release the occlusive pressure on the ulnar artery only to determine whether the modified allen test is positive or negative
- 4. if the hand flushes within 5-15 seconds it indicates that the ulnar artery has good flow and blood sample can be taken for test.

Screen baseline blood glucose, urine samples will be taken for screening baseline urine sugar and urine ketone. Patients will be assessed for blood sugar, urine ketone, urine sugar. Urine routine and blood routine was done.

STATISTICAL ANALYSIS:

All the dates will be entered in a data collection sheet in an Excel format and analysed using SPSS Software. Numerical values will be reported using mean and standard deviation or median. Categorical values will be reported using number and percentages. Probability value (p) value less than 0.05 was considered a statistically significant.

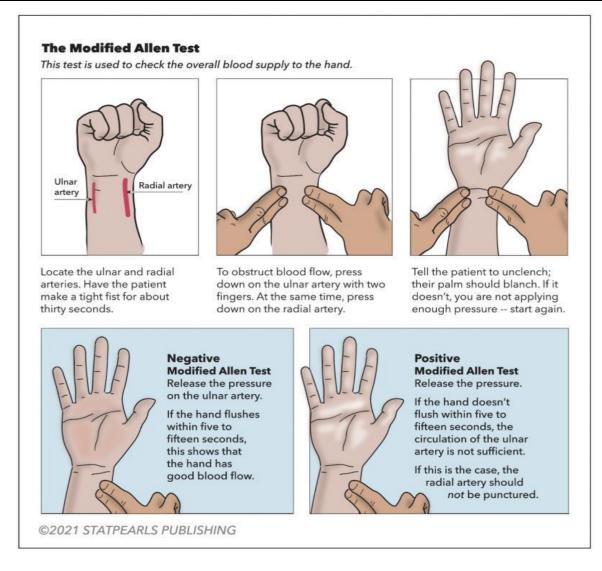
Specimen Requirements and Procedure

Whole blood is the required specimen for an arterial blood gas sample. The specimen is obtained through an arterial puncture or acquired from an indwelling arterial catheter. Once obtained, the arterial blood sample should be placed on ice and analyzed as soon as possible to reduce the possibility of erroneous results. Automated blood gas analyzers are commonly used to analyze blood gas samples, and results are obtained within 10 to 15 minutes. Automated blood gas analyzers, directly and indirectly, measure specific components of the arterial blood gas sample (see above). ABG Components:

- pH = *measured* acid-base balance of the blood
- PaO2 = *measured the* partial pressure of oxygen in arterial blood
- PaCO2 = *measured* the partial pressure of carbon dioxide in arterial blood
- HCO3 = *calculated* concentration of bicarbonate in arterial blood
- Base excess/deficit = calculated relative excess or deficit of base in arterial blood
- SaO2 = *calculated* arterial oxygen saturation unless a co-oximetry is obtained, in which case it is *measured*

Testing Procedures

A modified Allen test is a must before an ABG is drawn from either of the upper extremities to check for adequate collateral flow. Alternatively, pulse oximetry and duplex ultrasound can be used too. The arterial site commonly used is the radial artery, as it is superficial and easily palpable over the radial styloid process. The next most common site is the femoral artery.



The test is performed on the unilateral upper extremity chosen for the procedure (Please look at the attached image for graphical illustration). The selected upper extremity is flexed at the elbow, and the patient requested to clench the raised fist for 30 seconds. Then pressure is applied over the ulnar and radial arteries with the intent to occlude the blood flow. After five seconds, unclench the raised fist. The palm will now appear pale, white, or blanched. Then pressure over the ulnar artery is released while the radial artery compression is maintained. In 10 to 15 seconds, the palm returns to its original color, indicating adequate Ulnar collateral blood flow. If the palm does not return to its actual color, it is an abnormal test and unsafe to puncture the radial artery. Similarly, the radial collateral blood flow is assessed by maintaining ulnar artery pressure and releasing the radial artery pressure.

Results, Reporting, Critical Findings

An acceptable normal range of ABG values of ABG components are the following, noting that the range of normal values may vary among laboratories and in different age groups from neonates to geriatrics:

- pH (7.35-7.45)
- PaO2 (75-100 mmHg)
- PaCO2 (35-45 mmHg)
- HCO3 (22-26 meg/L)
- Base excess/deficit (-4 to +2)
- SaO2 (95-100%)

Arterial blood gas interpretation is best approached systematically. Interpretation leads to an

understanding of the degree or severity of abnormalities, whether the abnormalities are acute or chronic, and if the primary disorder is metabolic or respiratory in origin. Several articles have described simplistic ways to interpret ABG results.

The first step is to look at the pH and assess for the presence of acidemia (pH < 7.35) or alkalemia (pH > 7.45). If the pH is in the normal range (7.35-7.45), use a pH of 7.40 as a cutoff point. In other words, a pH of 7.37 would be categorized as acidosis, and a pH of 7.42 would be categorized as alkalemia. Next, evaluate the respiratory and metabolic components of the ABG results, the PaCO2 and HCO3, respectively. The PaCO2 indicates whether the acidosis or alkalemia is primarily from a respiratory or metabolic acidosis/alkalosis. PaCO2 > 40 with a pH < 7.4 indicates a respiratory acidosis, while PaCO2 < 40 and pH > 7.4 indicates a respiratory alkalosis (but is often from hyperventilation from anxiety or compensation for a metabolic acidosis). Next, assess for evidence of compensation for the primary acidosis or alkalosis by looking for the value (PaCO2 or HCO3) that is not consistent with the pH. Lastly, assess the PaO2 for any abnormalities in oxygenation.

When evaluating a patient's acid-base status, it is important to include an electrolyte imbalance or anion gap in your synthesis of the information. For example: In a patient who presents with Diabetic Ketoacidosis, they will eliminate ketones, close the anion gap but have persistent metabolic acidosis due to hyperchloremia. This is due to the strong ionic effect, which is beyond the scope of this article.

Clinical Significance

Arterial blood gas monitoring is the standard for assessing a patient's oxygenation, ventilation, and acid-base status. Although ABG monitoring has been replaced mainly by non-invasive monitoring, it is still useful in confirming and calibrating non-invasive monitoring techniques.

Acid-base balance can be affected by the respiratory system abnormalities. For instance, acute respiratory acidosis and alkalemia result in acidemia and alkalemia, respectively. Additionally, hypoxemic hypoxia leads to anaerobic metabolism, which causes metabolic acidosis that results in acidemia. Metabolic system abnormalities also affect acid-balance as acute metabolic acidosis and alkalosis result in acidemia and alkalemia, respectively. Metabolic acidosis is seen in patients with diabetic ketoacidosis, septic shock, renal failure, drug or toxin ingestion, and gastrointestinal or renal HCO3 loss. Metabolic alkalosis is caused by conditions such as kidney disease, electrolyte imbalances, prolonged vomiting, hypovolemia, diuretic use, and hypokalemia.

OBSERVATIONS AND RESULTS

Table 1 Age wise distribution of study participants

		J
Age Group	Frequency	%
≤ 20 Year	16	16.5%
21-40 Year	33	34%
41-60 year	34	35.1%
>60 Year	14	14.4%
Age (Mean \pm SD)	40.53 ± 17.47	

TABLE -1 Shows the demographic profile of patients with DKA. 16.5% of patients were between the age group of <20 years. 34% of them were between 21-40 years, 35.1% of them were between 41-60 years, 14.4% of them were > 60 years . **Mean Age+/- SD becomes 40.53+/-17.47**

Table 2 Gender wise distribution of study participants

		<i>.</i> 1
Age Group	Frequency	%
Male	70	72.2%
Female	27	27.8%
Total	97	100%

Table 2 showing gender wise distribution of study participants comprising 72.2% male patients and 27.8% Female patients.

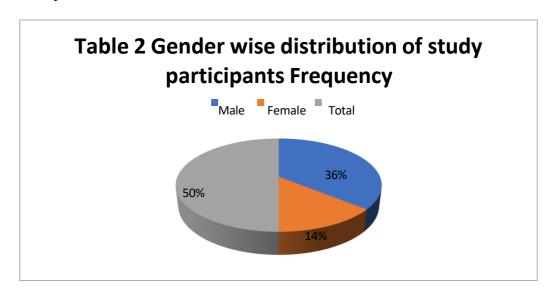


Table 3 Presenting Complaints of study participants

Presenting Complaints	Frequency	%
Missed abdominal pain	22	22.7%
Altered sensorium	24	24.7%
Dizziness	6	6.2%
Shortness of breath	17	17.5%
Vomiting	28	28.9%
Total	97	100%

Table No 3 showing distribution of patients according to presenting Complaints with 28.9% patients presented with vomiting , 22.7% presented with abdominal pain , 24.7% presented with altered sensorium, 17.5% presented with Shortness of breath.

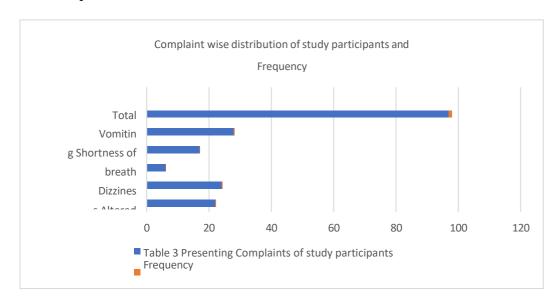


Table 4 Type of Diabetes among study participants

		/ <u>1</u>
Type of Diabetes	Frequency	%
Type 1 DM	42	43.3%
Type 2 DM	55	56.7%
Total	97	100%

Table 4 showing 56.7% patients presented with Type 2 diabetes mellitus while 43.3% presented with Type 1 Diabetes Mellitus

Table 5 Associated illnesses among study participants

	<i>U</i>	<i>J</i> 1 1
Associated illnesses	Frequency	%
CAD	4	4.1%
CVA	1	1%
HTN	16	16.5%
HTN/Hypothyroidism	1	1%
HTN/CAD	3	3.1%
Hypothyroidism	2	2.1%
No	70	72.2%
Total	97	100%

Table 5 shows 16.5% patients had preexisting hypertension, 4.1% has CAD, 3.1% has both hypertension and CAD 2.1% has Hypothyroidism rest 72.2% has no preexisting illness

Table 6 ECG changes among study participants

ECG changes	Frequency	%
Normal study	40	41.2%
Sinus Tachycardia	49	50.5%
Tallt waves (V1-V5)	8	8.2%
Total	9	100%

Table 6 Almost half (50.5%) of the participants were showing Sinus tachycardia, 41.2% showed normal ECG study while 8.2% showed significant ECG changes with Tall T waves in precordial leads.

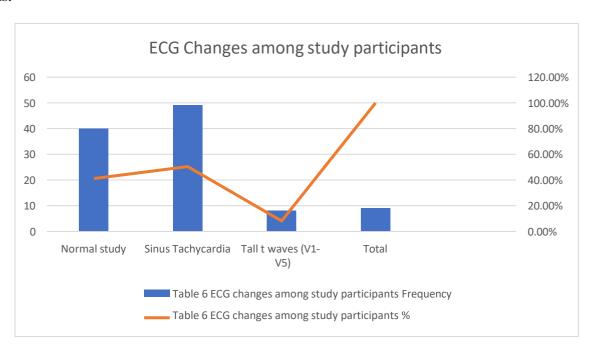


Table 7 outcome of study participants

		<i>y</i> 1
Outcome	Frequency	%
Expired	19	19.6%
Recovered	78	80.4%
Total	97	100%

Table 7 showing 80.4% out of all study participants recovered while 19.6% expired

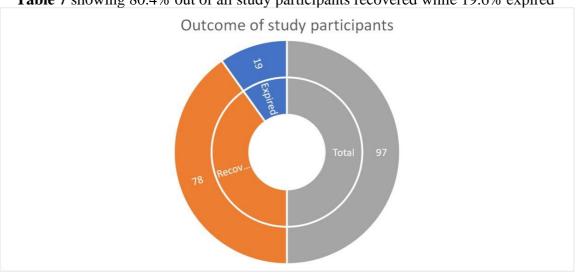


Table 8 duration of illnesses

Tuble o duration of innesses					
Duration of illnesses Frequency %					
<1 Year	3	3.1%			
1-5Year	47	48.5%			
6-10 year	29	29.9%			
11-15 Year	14	14.4%			
>15 year	4	4.1%			

Table 8 shows 48.5% patients presented in DKA has duration of illnesses 1-5 year, 29.9% has duration of illnesses 6-10year, 14.4% has duration of illness 14.4%, while 4.1% patients had duration of illnesses >15 years

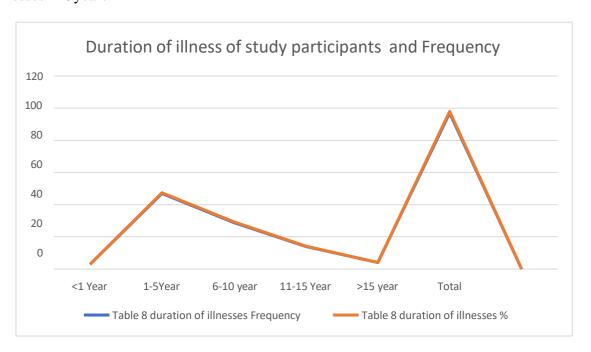


Table 10 Comparison between arterial and venous Blood Gas analysis variables

Blood Gas Analysis	Arterial	Venous	p Value
pН	7.25±0.07	7.25±0.06	0.65
HCO3	17.39±3.05	18.36±3.02	0.027
PO2	76.95±8.91	36.98±7.33	< 0.001
PCO2	39.76±3.4	60.65±10.63	< 0.001
Na+	134.32±1.96	134.71±2.67	0.245
K+	4.23±0.59	3.91±0.59	< 0.001

Table10 shows Metabolic Acidosis in both Arterial and Venous Blood sample.

Table 11 Comparison between arterial and venous Blood Gas analysis variables among expired study participants

Blood Gas Analysis	Arterial	Venous	p Value
pН	7.24±0.07	7.26±0.06	0.543
HCO3	18.74±2.73	19.58±2.67	0.343
PO2	80.26±9.61	35.47±6.16	< 0.001
PCO2	40.68±3.9	57.16±10.28	< 0.001
Na+	134.58±1.64	134.89±1.82	0.578
K+	3.93±0.5	4.04±0.6	0.569

Table 12 Comparison between arterial and venous Blood Gas analysis variables among recovered study participants

puritivi puriti			
Blood Gas Analysis	Arterial	Venous	p Value
рН	7.25±0.07	7.25±0.06	0.843
HCO3	17.07±3.06	18.07±3.04	0.042
PO2	76.14±8.6	37.35±7.58	< 0.001
PCO2	39.54±3.26	61.5±10.6	< 0.001
Na+	134.25±2.03	134.66±2.84	0.301
K+	4.3±0.59	3.87±0.59	< 0.001

Table 13 Comparison between expired and recovered study participants

Blood Gas Analysis Expired Recovered p Value				
	64.89±11.33	34.59±12.98	<0.001	
Age				
RBS	414.79±90.39	416.13±84.79	0.952	
Arterial pH	7.24±0.07	7.25±0.07	0.621	
Arterial HCO3	18.74±2.73	17.07±3.06	0.032	
Arterial PO2	80.26±9.61	76.14±8.6	0.070	
Arterial PCO2	40.68±3.9	39.54±3.26	0.190	
Arterial Na+	134.58±1.64	134.25±2.03	0.519	
Arterial K+	3.93±0.5	4.3±0.59	0.014	
VenouspH	7.26±0.06	7.25±0.06	0.868	
VenousHCO3	19.58±2.67	18.07±3.04	0.050	
Venous PO2	35.47±6.16	37.35±7.58	0.320	
Venous PCO2	57.16±10.28	61.5±10.6	0.111	
Venous Na+	134.89±1.82	134.66±2.84	0.737	
Venous K+	4.04±0.6	3.87±0.59	0.283	
Serum Urea	33.53±5.77	34.26±9.52	0.750	
Serum Creatinine	0.93±0.37	0.97±0.34	0.588	

Table13 Comparison between expired and recovered study participants

Blood Gas Analysis	Type 1 DM	Type 2 DM	P Value
Age	24.17±7.01	53.02±11.77	< 0.001
RBS	402±88.7	426.45±82.08	0.164
Arterial pH	7.24 ± 0.07	7.26±0.06	0.310

Arterial HCO3	16.88±3.32	17.79±2.8	0.149
Arterial PO2	76.07±8.59	77.62±9.17	0.400
Arterial PCO2	39.57±3.34	39.91±3.47	0.631
Arterial Na+	133.96±2.06	134.59±1.85	0.114
Arterial K+	4.25±0.56	4.21±0.62	0.731
Venous pH	7.25±0.06	7.26±0.06	0.434
VenousHCO3	17.88±3.29	18.73±2.77	0.171
VenousPO2	37.33±7.89	36.71±6.94	0.680
VenousPCO2	62.67±9.85	59.11±11.02	0.103
Venous Na+	134.34±2.55	134.99±2.74	0.233
Venous K+	3.89±0.55	3.92±0.63	0.786
Serum Urea	32.33±8.72	35.47±8.85	0.085
Serum Creatinine	1.03±0.35	0.91±0.33	0.087

DISCUSSION

In this study 'TO STUDY THE CORRELATION BETWEEN ARTERIAL AND VENOUS BLOOD GAS ANALAYSIS IN PATIENTS IN DIABETIC KETOACIDOSIS WITH REFRENCE TO OUTCOME' study was done on 97 Diabetic patients admitted in medical ward, emergency, and intensive care unit of medicine department of G. R. Medical college and JAH group of hospitals gwalior.

In total of 97 DKA patients studied 19.6% of them had unfavorable outcome. This was near similar to the studies conducted by Oschatz et al.165 and Agarwal A et al.9 who showed 29% and 30% mortality in their study in DKA patients. The mortality is rarely due to metabolic complications and is mostly due to underlying precipitating illness. The deaths in study population showed that the individuals who died had, one or other of the accompanied co-morbid illness, or complicated precipitating factor.

In this study, the median age of incidence of DKA was 40.53 ± 17.47 years, with increased incidence in ages less than 40 years of age, and age was not found significant statistically, affecting the outcome. This study was similar to results obtained by Agarwal et al who included a total of 270 patients in the study.9 In international study published in ADA,3stated that most patients with DKA were between the ages of 18 and 44 years (56%) and 45 and 65 years (24%), with only 18% of patients <20 years of age;3 and the prognosis of DKA is worsened with increasing age.

Slighlty higher mean age were seen in study of **Sandeep K. Immadisetty and Aparna P. Patange** with mean age of 56 years with 41% being above 60 years.

Our study shown male predilection in sex with total of 70 males and 27 females. Females have found to have increased incidence of DKA, whereas Agarwal A et al showed male gender had 7.93 fold more favorable outcome.

This finding was more concurrent with a study by Elleman et al., (1984) whereas it was contrary to the findings of Lee et al., (1987) who reported that DKA is more common in females.

In previous studies that two-thirds of diabetic patients presenting with DKA are Type 1 DM and DKA related to Type 2DM has an intractable course and worst outcomes. In this study, patients with Type 1 DM was 43.3% whereas of Type 2 DM were 56.7%. A similar result was produced by **Adhikari et al.**, which showed 62.8% of DKA patients had T2DM compared to 37.8% with Type 1DM .The reason for increased incidence of DKA in Type 2DM can be an indicator of changing profile in Type 2DM due to influence of changing social and environmental factors in developing countries like India, which is required to be scrutinized .DKA mainly occurs in patients with type 1 DM because these patients present with a complete lack of insulin that inhibits gluconeogenesis and glycogenolysis in insulin resistant states (Type 2 DM), the body remains sensitive to the anti-lipolytic effects of insulin. Thus, patients with type 2 DM are rarely affected (**Barski et al., 2013; Puttanna et al., 2014**)). However, this finding has been challenged in larger number of patients with type 2 DM presenting with DKA. This was seen in a study by **Balasubramanian et al., (1999)** who reported that 39% of the patients with DKA in their study had Type 2 DM (**Balasubramanyam et al., 1999**).

In our study 28.9% patient in DKA presented mainly with c/o vomiting, 24.7% in altered sensorium

and 17.5% with c/o shortness of breath In study by **Otieno et al.,173** altered level of consciousness was a major predictor of mortality in DKA patients whereas **Agarwal et al.** did not show such association.

Among the studied 97patients, most common co-morbid condition was hypertension (16.5%) followed by CAD (4.1%) and Hypothyroidism (2.1%). Hypertension was found in 86 % of CKD patients in study by Rajnikumari et al^[93], and 80% in Ridao et al^[99]. Similarly, Shafi S et al^[100]reported that among CKD patient, 84.8% had hypertension and 7% had diabetes mellitus. In study of Andrew DePiero, MD, Nathan Kuppermannn, MD and PECARN DKA FLUID STUDY GROUP Many patients presented in DKA present with hypertension or develop hypertension during treatment. In this study, we documented an association between hypertension and more severe acidosis and hypocapnia (pH and pCO2). Furthermore, we found that hypertension during DKA treatment was associated with alterations in mental status, even after adjusting for factors reflecting DKA severity. Although the number of patients with clinically overt cerebral injury in the study (~1%) was small, precluding meaningful analysis, the frequency of clinically overt cerebral injury in hypertensive patients was higher than in patients without hypertension. Hypertension at presentation was not significantly associated with GCS abnormalities in multivariable models, however, this may have reflected delayed manifestation of mental status In our study 41.2% among study population showed no ECG changes while 50.5% shown sinus tachycardia and 8.2% shown significant ECG changes with Tall T waves in precordial leads but according to Deniz Aygün, Fatih Aygün, [...], and Agop Çıtak many patients in DKA shown arrythmia ,acute myocardial infarction changes. Acute cardiac complications observed in diabetic ketoacidosis have generally been attributed to electrolyte imbalance. Hypokalemia, hyperkalemia, hypocalcemia, hypophosphatemia, and hypomagnesemia may develop in diabetic ketoacidosis. Hypokalemia is the most commonly observed electrolyte imbalance and most frequently leads to fatal arrhythmia. Hypokalemia at the time of diagnosis was observed in only four of our patients and the severity of hypokalemia was low. Ventricular premature beat on ECG was found in only one patient who had hypokalemia. ECG assessments were found to be normal in four children who were found to have hypokalemia and in four children whose corrected sodium level was found to be low.

In our study mean Arterial pH were 7.24 ± 0.07 , mean arterial bicarbonates were 18.74 ± 2.73 while mean venous pH was 7.26 ± 0.06 and mean venous bicarbonates were 19.58 ± 2.67 in expired patients. Similarly mean Arterial pH were 7.25 ± 0.07 , mean arterial bicarbonates were 17.07 ± 3.06 while mean venous pH was 7.25 ± 0.06 and mean venous bicarbonates were 18.07 ± 3.04 in recovered patients. According to study of

G. Balaji1, R. Devi Richarasanov SK, et al. also found that arterial venous pH, Bicarbonate levels were strongly correlated and they concluded that venous blood gas samples were a reliable indicator in patients with DKA and so venous puncture is an easy procedure compared to repeated arterial puncture. **Hatice Dulber G, et al.** in their study showed the mean difference between arterial and venous bicarbonate level is 1.88±

0.4. Similarly, our study showed the mean difference between arterial and venous bicarbonate level of 1.7 ± 0.5 [13]. Similarly in our study also showed that 88.5% patient has admitted ICU, 7% patients were admitted in the emergency ward.

SUMMARY

The present study "To Study The Correlation Between Arterial And Venous Blood Gas Analysis In Patients In Diabetic Ketoacidosis With Refrence To OUTCOME" has been conducted in Department of Medicine, G.R. Medical College and J.A. Group of Hospitals, Gwalior (M.P.). A total of 97 cases were studied from January 2021 to November 2022.

- -Among the studied 97 patients, maximum cases belonged to the age group 41-60 years (n=34). Mean age of the study population was 40.53 years with standard deviation of 17.47 years.
- -Out of studied 97 patients, 70 were male and 27 were female.
- -In our study, most common clinical symptom was vomiting (found in 28.9% patients), followed by altered sensorium (24.7% each), while abdominal pain 22.7% and shortness of breath was found in

17.5 % patients respectively.

- -Among the studied 97 patients,16 were having associated hypertension and 4 patients were having CAD, while only 2 patients were suffering from hypothyroidism.
- -In our study, Type 1 DM patients were 43.3% while 56.7% were Type 2 DM.
- -Among the patients 41.2% shown no ECG changes, while 50.5% shown sinus tachycardia and only 8.2% shown significant ECG changes with tall T waves in precordial leads.
- -Among the studied 97 patients, 19.6% expired while 80.4% recovered.
- -The mean value of Arterial pH is 7.24±0.07 and Arterial bicarbonates is 18.74±2.73. while mean Venous pH is 7.26±0.06 and Venous Bicarbonates is 19.58±2.67 in expired patients.
- -The mean value of Arterial pH is 7.25 ± 0.07 and Arterial bicarbonates is 17.07 ± 3.06 while mean Venous pH is 7.25 ± 0.06 and Venous Bicarbonates is 18.07 ± 3.04 in expired patients.

CONCLUSION

Present study comprises of 96 patients of Diabetes Mellitus (newly diagnosed and known cases).

It was concluded that venous blood gas analysis has got advantages over arterial blood gas analysis like safety, fewer number of punctures, easy sampling, less painful, less invasive even though there are some reservations' analysis safer alternative to ABG for determining acid base status reducing the need for frequent invasive arterial sampling.

This study suggests that VBG pH values very closely correlate with ABG pH values, which also shows VBG substitution for ABG.

Hence venous blood gas might be used as an ideal alternative to arterial blood gas in the initial management of patients in Diabetic Ketoacidosis.

REFERENCES

- 1. Zisquit J, Velasquez J, Nedeff N. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Apr 30, 2022. Allen Test. [PubMed]
- 2. Cowley NJ, Owen A, Bion JF. Interpreting arterial blood gas results. BMJ. 2013 Jan 16;346:f16. [PubMed]
- 3. Larkin BG, Zimmanck RJ. Interpreting Arterial Blood Gases Successfully. AORN J.2015 Oct; 102 (4):343-54; quiz 355-7. [PubMed]
- 4. Rogers KM, McCutcheon K. Four steps to interpreting arterial blood gases. J PerioperPract. 2015 Mar;25(3):46-52. [PubMed]
- 5. Romanski SO. Interpreting ABGs in four easy steps (continuing education credit). Nursing. 1986 Sep;16(9):58-64. [PubMed]
- 6. ARDS Definition Task Force. Ranieri VM, Rubenfeld GD, Thompson BT, Ferguson ND, Caldwell E, Fan E, Camporota L, Slutsky AS. Acute respiratory distress syndrome: the Berlin Definition. JAMA. 2012 Jun 20;307(23):2526-33. [PubMed]
- 7. Pediatric Acute Lung Injury Consensus Conference Group. Pediatric acute respiratory distress syndrome: consensus recommendations from the Pediatric Acute Lung Injury Consensus Conference. PediatrCrit Care Med. 2015 Jun;16(5):428-39. [PMC free article] [PubMed]
- 8. Rawat M, Chandrasekharan PK, Williams A, Gugino S, Koenigsknecht C, Swartz D, Ma CX, Mathew B, Nair J, Lakshminrusimha S. Oxygen saturation index and severity of hypoxic respiratory failure. Neonatology. 2015;107(3):161-6. [PMC free article] [PubMed]
- 9. Kurt OK, Alpar S, Sipit T, Guven SF, Erturk H, Demirel MK, Korkmaz M, Hayran M, Kurt B. The diagnostic role of capnography in pulmonary embolism. Am J Emerg Med. 2010 May;28(4):460-[PubMed]
- 10. Davis MD, Walsh BK, Sittig SE, Restrepo RD. AARC clinical practice guideline: blood gas analysis and hemoximetry: 2013. Respir Care. 2013 Oct;58(10):1694-703. [PubMed]
- 11. Albert TJ, Swenson ER. Circumstances When Arterial Blood Gas Analysis Can Lead Us Astray. Respir Care. 2016 Jan;61(1):119-21
- 12. Muakkassa FF, Rutledge R, Fakhry SM, Meyer AA, Sheldon GF. ABGs and arterial lines: The relationship to unnecessarily drawn arterial blood gas samples. J Trauma. 1990;30:1087–

- 13. 93. [PubMed] [Google Scholar]
- 14. Guidelines for standards of care of patients with acute respiratory failure on mechanical ventilator support. Task Force on Guidelines; Society of Critical Care Medicine. Crit Care Med. 1991;19:275–8. [PubMed] [Google Scholar]
- 15. Barker WJ. Arterial puncture and cannulation. In: Roberts JR, Hedges JR, editors. Clinical Procedure in Emergency Medicine. 3rd ed. Philadelphia: WB Saunders; 1998. pp. 308–22. [Google Scholar]
- 16. Malinoski DJ, et al. Correlation of central venous and arterial blood gas measurement in mechanically ventilated trauma patients. Arch Surg. 2005;140:122–5. [PubMed] [Google Scholar]
- 17. Kelly AM, McAlpine R, Kyle E. Agreement between bicarbonate measured on arterial and venous blood gases. Emerg Med Australas. 2004;16:407–9. [PubMed] [Google Scholar]
- 18. Ma OJ, Rush MD, Godfrey MM, Gaddis G. Arterial blood gas results rarely influence emergency physician management of patients with suspected diabetic ketoacidosis. Acad Emerg Med. 2003;10:836–41. [PubMed] [Google Scholar]
- 19. Gokel Y, Paydas S, Koseoglu Z, Alparslan N, Seydaoglu G. Comparison of blood gas and acid-base measurements in arterial and venous blood samples in patients with uremic acidosis and diabetic ketoacidosis in the emergency room. Am J Nephrol. 2000;20:319–[PubMed] [Google Scholar]
- 20. Kelly AM, McAlpine R, Kyle E. Venous pH can safely replace arterial pH in the initial evaluation of patients in the emergency department. Emerg Med J. 2001;18:340–2. [PMC free article] [PubMed] [Google Scholar]
- 21. Middleton P, Kelly AM, Brown J, Robertson M. Agreement between arterial and central venous values for pH, bicarbonate, base excess, and lactate. Emerg Med J. 2006;23:622–4. [PMC free article] [PubMed] [Google Scholar
- 22. Johnston HL, Murphy R. Agreement between an arterial blood gas analyser and a venous blood analyser in the measurement of potassium in patients in cardiac arrest. Emerg Med J. 2005;22:269–71. [PMC free article] [PubMed] [Google Scholar]
- 23. Kelly AM, Kyle E, McAlpine R. Venous pCO2 and pH can be used to screen for significant hypercarbia in emergency patients with acute respiratory disease. J Emerg Med. 2002;22:15–9. [PubMed] [Google Scholar]
- 24. Rang LC, Murray HE, Wells GA, Macgougan CK. Can peripheral venous blood gases replace arterial blood gases in emergency department
- 25. Balasubramanyam, A., Zern, JW., Hyman, DJ., Pavlik, V. (1999). New profiles of diabetic ketoacidosis: Type 1 vs type 2 diabetes and the effect of ethnicity. Archives of Internal Medicine. 159(19):2317
- 26. Barski, L., Nevzorov, R., Jotkowitz, A., Rabaev, E., Zektser, M., Zeller, L. (2013). Comparison of Diabetic Ketoacidosis in Patientswith Type-1 and Type-2 Diabetes Mellitus. The American Journal of the Medical Sciences. 345(4):326-330. Cross Ref
- 27. Brandenburg, MA., Dire, DJ. (1998). Comparison of arterial and venous blood gas values in the initial emergency department evaluation of patients with diabetic ketoacidosis. Annals of Emergency Medicine. 31(4):459–65. CrossRef
- 28. Brashear, RE., Oei, TO., Rhodes, ML., Futty, DE., Hostetler, ML. (1979). Relationship between arterial and venous bicarbonate values. Archives of Internal Medicine. 139(4):440–2. CrossRef
- 29. Cashen, K., Petersen, T. (2019). Diabetic Ketoacidosis. Pediatrics in Review. 40(8): 412-420. CrossRef
- 30. Ellemann, K., Soerensen, JN., Pedersen, L., Edsberg, B., Andersen, OO. (1984). Epidemiology and treatment of diabetic ketoacidosis in a community population. Diabetes Care. 7(6):528–32. CrossRef
- 31. Farsani, SF., Brodovicz, K., Soleymanlou, N., Marquard, J., Wissinger, E., Mayes, BA. (2017). Incidence and prevalence of diabetic ketoacidosis (DKA) among adults with type 1 diabetes mellitus (T1D): a systematic literature review. BMJ Open. 7:e016587. CrossRef

- 32. Ganie, MA., Koul, S., Razvi, HA., Laway, BA., Zargar, AH. (2012). Hyperglycemic emergencies in Indian patients with diabetes mellitus on pilgrimage to Amarnathjiyatra. Indian Journal of Endocrinology and Metabolism. 16 (Suppl 1):S87–90. Cross Ref
- 33. Gokel, Y., Paydas, S., Koseoglu, Z., Alparslan, N., Seydaoglu, G. (2000). Comparison of Blood Gas and Acid-Base Measurements in Arterial and Venous Blood Samples in Patients with Uremic Acidosis and Diabetic Ketoacidosis in the Emergency Room. American Journal of Nephrology. 20(4):319–323. CrossRef
- 34. Jameson, JL., Fauci, AS., Kasper, DL., Hauser, SL., Longo, DL., Loscalzo, J. (2018) Harrison's principles of Internal Medicine. 20th ed. Shanahan JF, Davis KJ, editors. United States of America: McGraw-Hill Education: p2870–2872.
- 35. Kelly, A-M. (2006). The case for venous rather than arterial blood gases in diabetic ketoacidosis. Emergency Medicine Australas. 18(1):64–7. CrossRef
- 36. Kitabchi, AE., Wall, BM. (1995). Diabetic ketoacidosis. Medical Clinics of North America. 79(1):9–37. CrossRef
- 37. Lee, HK., Oh, YS., Chung, YH., Yoo, HJ., et al. (1987).
- 38. Epidemiological Characteristics of Ketoacidosis among Korean Diabetic patients. Journal of Korean Medical Science. 2(1):7–11. CrossRef
- 39. Lwanga, S., Lemeshow, S. (1991). Sample Size Determination in Health Studies. Geneva: World Health Organization. 1-30.
- 40. Mortensen, J. (1967). Clinical Sequelae from Arterial Needle Puncture, Cannulation, and Incision. Circulation. 35(6):1118–23. CrossRef
- 41. Puttanna, A., Padinjakara, RNK. (2014). Diabetic ketoacidosis in type 2 diabetes mellitus. Practical Diabetes. 31(4): 155–158. <u>CrossRef</u>
- 42. Roberts, JR., Custalow, CB., Thomsen, TW. (2017). Roberts and Hedges clinical procedures in emergency medicine and acute care. 7th Edition. Elsevier Saunders. p1541.Singh, V., Khatana, S., Gupta, P. (2013). Blood gas analysis for bedside diagnosis. National Journal of Maxillofacial 4(2):136–41. CrossRef
- 43. Trachtenbarg, DE. (2005). Diabetic Ketoacidosis. American Family Physician. 71(9):1705–14.
- 44. 21) Bissler J, Welch T, Loggie J Paradoxical hypertension in hypovolemic children. Pediatric Emergency Care 1991; 7: 350–2. [PubMed] [Google Scholar]
- 45. Glaser NS, Wootton-Gorges SL, Marcin JP, Buonocore MH, Dicarlo J, Neely EK et al. Mechanism of cerebral edema in children with diabetic ketoacidosis. J Pediatr. 2004;145:164–71. [PubMed] [Google Scholar]
- 46. Richarasanov SK, M.D. Dire DJ. Comparison of arterial and venous blood gas values in the initial emergency department evaluation of patients with diabetic ketoacidosis. Ann Emerg Med., April 2002; 31: 459-65.
- 47. Hatice Dulber G, lruPolat Melih B.Y. Comparison of Arterial and Venous Blood Gas Values in case of COPD with acute respiratory failure. 2004; 8(9): 936-944.
- 48. Richard Treger, Shariar Pirouz, Nader Kamagra. Compared central venous and arterial blood gas measurements in the ICU. 2004; 16: 407- 409.